What is claimed:

1. A method of therapeutically treating an individual afflicted with Alzheimer's disease comprising administering a compound to the individual which inhibits amyloid precursor protein phosphorylation in a manner to reduce, prevent, or reverse symptoms associated with Alzheimer's disease.

2. The method of claim 1, wherein a kinase is inhibited.

3. The method of claim 2, wherein the kinase is prevented from phosphorylating an amino acid residue of the amyloid precursor protein.

4. The method of claim 3, wherein the amino acid residue is selected from the group consisting of tyrosine 653, serine 655, threonine 668, serine 675, tyrosine 682, threonine 686 and tyrosine 687.

5. The method of claim 1, wherein hyperphosphorylation of the amyloid precursor protein is inhibited.

6. The method of claim 1, wherein the compound is a cyclin-dependent kinase inhibitor.

7. The method of claim 6, wherein the compound is selected from the group consisting of butyrolactone, roscovitine, olomoucine, kenpaullone, and alsterpaullone.

8. The method of claim 2, wherein the kinase is a cyclin dependent kinase.

9. The method of claim 8, wherein the cyclin dependent kinase is cyclin dependent kinase 5.

10. The method of claim 1, wherein the amyloid precursor protein is a carboxyl terminal fragment of amyloid precursor protein.

11. The method of claim 1, wherein the symptoms are selected from the group consisting of behavioral symptoms, physical symptoms, and pathological symptoms.

12. A method of inhibiting amyloid precursor protein phosphorylation in an individual comprising contacting an individual with a compound, wherein the compound inhibits phosphorylation of the amyloid precursor protein.

13. The method of claim 12, wherein activation of a kinase is inhibited.

14. The method of claim 13, wherein the kinase is inhibited from phosphorylating an amino acid residue of the amyloid precursor protein.

15. The method of claim 14, wherein the amino acid residue is selected from the group consisting of tyrosine 653, serine 655, threonine 668, serine 675, tyrosine 682, threonine 686 and tyrosine 687.

16. The method of claim 12, wherein hyperphosphorylation of the amyloid precursor protein is inhibited.

17. The method of claim 12, wherein the compound is a cyclin-dependent kinase inhibitor.

18. The method of claim 17, wherein the compound is selected from the group consisting of butyrolactone, roscovitine, olomoucine, kenpaullone, and alsterpaullone.

19. The method of claim 12, wherein the kinase is a cyclin-dependent kinase.

20. The method of claim 19, wherein the cyclin dependent kinase is cyclin dependent kinase 5.

21. The method of claim 12, wherein the amyloid precursor protein is a carboxyl terminal fragment of amyloid precursor protein.

22. The method of claim 12, wherein the individual is afflicted with Alzheimer's disease.

23. A method of diagnosing Alzheimer's disease in a patient comprising:

(a) obtaining a biological sample from a patient;

(b) obtaining a biological sample from an individual without Alzheimer's disease;

(c) comparing amyloid precursor protein phosphorylation in the biological sample from the patient to amyloid precursor protein phosphorylation in the biological sample from an individual without Alzheimer's disease; and

(d) diagnosing the patient with Alzheimer's disease when the biological sample from the patient has increased amyloid precursor protein phosphorylation compared to amyloid precursor protein phosphorylation in the biological sample from the individual without Alzheimer's disease.

24. The method of claim 23, wherein the biological sample is selected from the group consisting of spinal tissue, brain tissue, cerebrospinal fluid, blood, lymph, sputum, and urine.

25. The method of claim 23, wherein the phosphorylation occurs at an amino acid residue of the amyloid precursor protein selected from the group consisting of tyrosine 653, serine 655, threonine 668, serine 675, tyrosine 682, threonine 686 and tyrosine 687.

26. The method of claim 23, wherein the phosphorylation is hyperphosphorylation.

27. The method of claim 23, wherein the phosphorylation of amyloid precursor protein is determined by an antibody that binds a phosphorylated form of amyloid precursor protein.

28. A method of diagnosing Alzheimer's disease in a patient comprising:

(a) obtaining a biological sample from a patient;

(b) comparing amyloid precursor protein phosphorylation in the biological sample from the patient to a database comprising amyloid precursor protein phosphorylation data from samples taken from subjects at various stages of Alzheimer's disease; and

(c) diagnosing the patient with Alzheimer's disease when the biological sample from the patient has amyloid precursor protein phosphorylation that is the same or increased compared with amyloid precursor protein phosphorylation data from a subject in the database.

29. The method of claim 28, wherein the biological sample is selected from the group consisting of spinal tissue, brain tissue, cerebrospinal fluid, blood, lymph, sputum, and urine.

30. The method of claim 28, wherein the phosphorylation occurs at an amino acid residue of the amyloid precursor protein selected from the group consisting of tyrosine 653, serine 655, threonine 668, serine 675, tyrosine 682, threonine 686 and tyrosine 687.

31. The method of claim 28, wherein the phosphorylation is hyperphosphorylation.

32. The method of claim 28, wherein the phosphorylation of amyloid precursor protein is determined by an antibody that binds a phosphorylated form of amyloid precursor protein.

33. A method of inhibiting cleavage of amyloid precursor protein, wherein phosphorylation of an amino acid residue of amyloid precursor protein is inhibited.

34. The method of claim 33, wherein beta-amyloid formation is inhibited.

35. The method of claim 34, wherein the beta-amyloid is beta-amyloid (1-40) or beta-amyloid (1-42).

36. The method of claim 35, wherein a kinase is inhibited.

37. The method of claim 36, wherein the kinase is a cyclin-dependent kinase.

38. The method of claim 37, wherein the cyclin-dependent kinase is cyclin dependent kinase 5.

39. The method of claim 36, wherein the kinase is inhibited by a compound selected from the group consisting of butyrolactone, roscovitine, olomoucine, kenpaullone, and alsterpaullone.

40. The method of claim 33, wherein a p25 activity is inhibited.

41. The method of claim 40, wherein the activity is activation of a kinase.

42. The method of claim 33, wherein a β-secretase activity is inhibited.

43. The method of claim 42, wherein the activity is cleavage of amyloid precursor protein.

44. The method of claim 33, wherein the amino acid residue is selected from the group consisting of tyrosine 653, serine 655, threonine 668, serine 675, tyrosine 682, threonine 686 and tyrosine 687.

45. A compound for inhibiting cleavage of amyloid precursor protein, wherein the compound inhibits phosphorylation of an amino acid residue of amyloid precursor protein.

46. The method of claim 45, wherein beta-amyloid formation is inhibited.

47. The method of claim 46, wherein the beta-amyloid is beta-amyloid (1-40) or beta-amyloid (1-42).

48. The method of claim 45, wherein a kinase is inhibited.

49. The method of claim 48, wherein the kinase is a cyclin-dependent kinase.

50. The method of claim 49, wherein the cyclin-dependent kinase is cyclin dependent kinase 5.

51. The method of claim 48, wherein the kinase is inhibited by a compound selected from the group consisting of butyrolactone, roscovitine, olomoucine, kenpaullone, and alsterpaullone.

52. The method of claim 45, wherein a p25 activity is inhibited.

53. The method of claim 52, wherein the activity is activation of a kinase.

54. The method of claim 45, wherein a β-secretase activity is inhibited.

55. The method of claim 54, wherein the activity is cleavage of amyloid precursor protein.

56. The method of claim 45, wherein the amino acid residue is selected from the group consisting of tyrosine 653, serine 655, threonine 668, serine 675, tyrosine 682, threonine 686 and tyrosine 687.

57. A method of identifying a compound that inhibits symptoms associated with Alzheimer's disease comprising:

(a) providing a sample comprising amyloid precursor protein and a kinase;

(b) dividing the sample into a first sample and a second sample;

(c) contacting the first sample with the compound; and

(d) determining phosphorylation of the amyloid precursor protein in the first and second samples, wherein a decrease of amyloid precursor protein phosphorylation is detected in the first sample relative to the second sample when the compound inhibits symptoms associated with Alzheimer's disease.

58. The method of claim 57, wherein the kinase is a cyclin-dependent kinase.

59. The method of claim 58, wherein the cyclin-dependent kinase is cyclin dependent kinase 5.

60. The method of claim 57, wherein the source of the kinase is a cell extract.

61. The method of claim 57, wherein the source of the kinase is a recombinant kinase.

62. The method of claim 57, wherein the phosphorylation occurs at an amino acid residue of the amyloid precursor protein selected from the group consisting of tyrosine 653, serine 655, threonine 668, serine 675, tyrosine 682, threonine 686 and tyrosine 687.

63. The method of claim 57, wherein phosphorylation of the amyloid precursor protein is detected using an antibody that binds phosphorylated amyloid precursor protein.

64. The method of claim 57, wherein phosphorylation of the amyloid precursor protein is detected using a kinase assay.

65. A method of identifying a compound that inhibits progression of Alzheimer's disease comprising:

(a) providing a sample comprising amyloid precursor protein and a kinase;

(b) dividing the sample into a first sample and a second sample;

(c) contacting the first sample with the compound; and

(d) determining phosphorylation of the amyloid precursor protein in the first and second samples, wherein a decrease of amyloid precursor protein phosphorylation is detected in the first sample relative to the second sample when the compound inhibits progression of Alzheimer's disease.

66. The method of claim 65, wherein the kinase is a cyclin-dependent kinase.

67. The method of claim 66, wherein the cyclin-dependent kinase is cyclin dependent kinase 5.

68. The method of claim 65, wherein the source of the kinase is a cell extract.

69. The method of claim 65, wherein the source of the kinase is a recombinant kinase.

70. The method of claim 65, wherein the phosphorylation occurs at an amino acid residue of the amyloid precursor protein selected from the group consisting of tyrosine 653, serine 655, threonine 668, serine 675, tyrosine 682, threonine 686 and tyrosine 687.

71. The method of claim 65, wherein phosphorylation of the amyloid precursor protein is detected using an antibody that binds phosphorylated amyloid precursor protein.

72. The method of claim 65, wherein phosphorylation of the amyloid precursor protein is detected using a kinase assay.

73. A transgenic mouse whose genome comprises a first transgene comprising a DNA sequence encoding p25 operably linked to an inducible promoter, and a second transgene comprising a DNA sequence encoding an inducer operably linked to a tissue-specific promoter.

74. The transgenic mouse of claim 73, wherein the inducible promoter is a tetracycline responsive element.

75. The transgenic mouse of claim 73, wherein the tissue-specific promoter is a calcium-calmodulin-dependent kinase II promoter.

76. The transgenic mouse of claim 73, wherein the inducer is a tetracycline-responsive transcriptional activator.

77. The transgenic mouse of claim 73, wherein the p25 is expressed in the brain.

78. The transgenic mouse of claim 73, wherein the p25 is expressed in the forebrain.

79. The transgenic mouse of claim 73, wherein the p25 is a murine p25.

80. The transgenic mouse of claim 73, wherein the p25 is a human p25.

81. The transgenic mouse of claim 73, wherein the transgenic mouse over-expresses p25 when compared to mouse not expressing the transgene comprising a DNA sequence encoding p25.

82. The transgenic mouse of claim 73, exhibiting one or more features selected from the group consisting of progressive neurodegeneration, tau aggregation, neurofibrillary tangle formation, aberrant cyclin-dependent kinase 5 activity, neuronal loss in the cerebral cortex, neuronal loss in the hippocampus, severe brain atrophy, reactive astrogliosis, caspase-3 activation, up-regulation of C99, up-regulation of beta-amyloid, tau hyperphosphorylation, amyloid precursor protein phosphorylation, and amyloid precursor protein hyperphosphorylation.

83. The transgenic mouse of claim 81, wherein the amyloid precursor protein phosphorylation or the amyloid precursor protein hyperphosphorylation occurs at one or more amino acid residues selected from the group consisting of tyrosine 653, serine 655, threonine 668, serine 675, tyrosine 682, threonine 686 and tyrosine 687.

84. The transgenic mouse of claim 73, exhibiting one or more behavioral symptoms of Alzheimer's disease.

85. A cell line established from the transgenic mouse of claim 73, wherein the cell line comprises a cell having a genome comprising a first transgene comprising a DNA sequence encoding p25 operably linked to an inducible promoter, and a second transgene comprising a DNA sequence encoding an inducer operably linked to a tissue-specific promoter.

86. An assay for determining the effect of a compound on a feature of a neurodegenerative disorder comprising:

a) providing a first transgenic mouse and a second transgenic mouse whose genomes comprise a first transgene comprising a DNA sequence encoding p25 operably linked to an inducible promoter, and a second transgene comprising a DNA sequence encoding an inducer operably linked to a tissue-specific promoter;

b) exposing the first transgenic mouse to a compound;

c) incubating the first transgenic mouse and the second transgenic mouse;

d) measuring the feature of a neurodegenerative disorder for the first transgenic mouse and the second transgenic mouse; and

e) comparing the measurements to determine the effect of the compound on the feature of the neurodegenerative disorder.

87. The assay of claim 86, wherein the neurodegenerative disorder is Alzheimer's disease.

88. The assay of claim 86, wherein the feature is a behavioral symptom of Alzheimer's disease.

89. The assay of claim 86, wherein the feature is selected from the group consisting of progressive neurodegeneration, tau aggregation, neurofibrillary tangle formation, aberrant cyclin-dependent kinase 5 activity, neuronal loss in the cerebral cortex, neuronal loss in the hippocampus, severe brain atrophy, reactive astrogliosis, caspase-3 activation, up-regulation of C99, up-regulation of beta-amyloid, tau hyperphosphorylation, amyloid precursor protein phosphorylation, and amyloid precursor protein hyperphosphorylation.

90. The assay of claim 89, wherein the amyloid precursor protein phosphorylation or the amyloid precursor protein hyperphosphorylation occurs at one or more amino acid residues selected from the group consisting of tyrosine 653, serine 655, threonine 668, serine 675, tyrosine 682, threonine 686 and tyrosine 687.

91. The assay of claim 86, wherein the compound is a cyclin-dependent kinase inhibitor.

92. The assay of claim 91, wherein the cyclin-dependent kinase inhibitor is selected from the group consisting of butyrolactone, roscovitine, olomoucine, kenpaullone, and alsterpaullone.

93. An assay for determining the effect of a compound on amyloid precursor protein phosphorylation comprising:

a) providing a first transgenic mouse and a second transgenic mouse whose genomes comprise a first transgene comprising a DNA sequence encoding p25 operably linked to an inducible promoter, and a second transgene comprising a DNA sequence encoding an inducer operably linked to a tissue-specific promoter;

b) exposing the first transgenic mouse to a compound;

c) incubating the first transgenic mouse and the second transgenic mouse;

d) measuring amyloid precursor protein phosphorylation in the first transgenic mouse and the second transgenic mouse; and

e) comparing the measurements to determine the effect of the compound on amyloid precursor protein phosphorylation.

94. An assay for determining the effect of a compound on a feature of a neurodegenerative disorder comprising:

a) providing a first cell and a second cell whose genomes comprise a first transgene comprising a DNA sequence encoding p25 operably linked to an inducible promoter, and a second transgene comprising a DNA sequence encoding an inducer operably linked to a tissue-specific promoter;

b) exposing the first cell to a compound;

c) incubating the first cell and the second cell;

d) measuring the feature of a neurodegenerative disorder in the first cell and the second cell; and

e) comparing the measurements to determine the effect of the compound on the feature of the neurodegenerative disorder.

95. The assay of claim 93, wherein the neurodegenerative disorder is Alzheimer's disease.

96. The assay of claim 94, wherein the feature is selected from the group consisting of tau aggregation, neurofibrillary tangle formation, aberrant cyclin-dependent kinase 5 activity, caspase-3 activation, up-regulation of C99, up-regulation of beta-amyloid, tau hyperphosphorylation, amyloid precursor protein phosphorylation, and amyloid precursor protein hyperphosphorylation.

97. The assay of claim 96, wherein the amyloid precursor protein phosphorylation or the amyloid precursor protein hyperphosphorylation occurs at one or more amino acid residues selected from the group consisting of tyrosine 653, serine 655, threonine 668, serine 675, tyrosine 682, threonine 686 and tyrosine 687.

98. The assay of claim 94, wherein the compound is a cyclin-dependent kinase inhibitor.

99. The assay of claim 98, wherein the cyclin-dependent kinase inhibitor is selected from the group consisting of butyrolactone, roscovitine, olomoucine, kenpaullone, and alsterpaullone.

100. An assay for determining the effect of a compound on amyloid precursor protein phosphorylation comprising:

a) providing a first and a second cell whose genomes comprise a first transgene comprising a DNA sequence encoding p25 operably linked to an inducible promoter, and a second transgene comprising a DNA sequence encoding an inducer operably linked to a tissue-specific promoter;

b) exposing the first cell to a compound;

c) incubating the first cell and the second cell;

d) measuring amyloid precursor protein phosphorylation in the first cell and the second cell; and

e) comparing the measurements to determine the effect of the compound on amyloid precursor protein phosphorylation.